Basolateral amygdala regulation of adult hippocampal neurogenesis and fear-related activation of newborn neurons.

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Public Summary:
Impaired regulation of emotional memory is a feature of several affective disorders, including depression, anxiety and post-traumatic stress disorder. Such regulation occurs, in part, by interactions between the hippocampus and the basolateral amygdala (BLA). Recent studies have indicated that within the adult hippocampus, newborn neurons may contribute to support emotional memory, and that regulation of hippocampal neurogenesis is implicated in depressive disorders. How emotional information affects newborn neurons in adults is not clear. Given the role of the BLA in hippocampus-dependent emotional memory, we investigated whether hippocampal neurogenesis was sensitive to emotional stimuli from the BLA. We show that BLA lesions suppress adult neurogenesis, while lesions of the central nucleus of the amygdala do not. Similarly, reducing BLA activity suppresses neurogenesis. We also show that BLA lesions prevent selective activation of immature newborn neurons in response to a fear-conditioning task. These results demonstrate that BLA activity regulates adult hippocampal neurogenesis and the fear context-specific activation of newborn neurons. Together, these findings denote functional implications for proliferation and recruitment of new neurons into emotional memory circuits.

Scientific Abstract:
Impaired regulation of emotional memory is a feature of several affective disorders, including depression, anxiety and post-traumatic stress disorder. Such regulation occurs, in part, by interactions between the hippocampus and the basolateral amygdala (BLA). Recent studies have indicated that within the adult hippocampus, newborn neurons may contribute to support emotional memory, and that regulation of hippocampal neurogenesis is implicated in depressive disorders. How emotional information affects newborn neurons in adults is not clear. Given the role of the BLA in hippocampus-dependent emotional memory, we investigated whether hippocampal neurogenesis was sensitive to emotional stimuli from the BLA. We show that BLA lesions suppress adult neurogenesis, while lesions of the central nucleus of the amygdala do not. Similarly, we show that reducing BLA activity through viral vector-mediated overexpression of an outwardly rectifying potassium channel suppresses neurogenesis. We also show that BLA lesions prevent selective activation of immature newborn neurons in response to a fear-conditioning task. These results demonstrate that BLA activity regulates adult hippocampal neurogenesis and the fear context-specific activation of newborn neurons. Together, these findings denote functional implications for proliferation and recruitment of new neurons into emotional memory circuits.